The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this monograph, the Agency for Toxic Substances and Disease Registry (ATSDR) has made a diligent effort to ensure the accuracy and currency of the information presented but makes no claim that the document comprehensively addresses all possible situations related to this substance. This monograph is intended as an additional resource for physicians and other health professionals in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider and must be interpreted in light of specific information regarding the patient available to such a professional and in conjunction with other sources of authority.

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Please send me the following Case Studies in Environmental Medicine. (Circle selected subjects.)

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CASE STUDIES IN ENVIRONMENTAL MEDICINE: ASBESTOS TOXICITY

If you wish CME credits or CEUs, please indicate your answers to the Posttest questions on the previous page by circling the letters below for the correct answers. Complete the evaluation questionnaire and fill in the information requested on the following page.

Mail your answer sheet, evaluation questionnaire and the information page which follows to: Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, NE, Atlanta, GA 30333.

1. a b c d e
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Evaluation Questionnaire

Please complete the following evaluation by circling the appropriate number.

	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
1. As a result of completing this monograph, I will be able to:					
Explain why asbestos may be an acute and chronic health hazard.	1	2	3	4	5
Describe the known factors contributing to asbestos poisoning.	1	2	3	4	5
Identify potential environmental or occupational sources of exposure to asbestos.	1	2	3	4	5
Identify evaluation and treatment protocols for persons exposed to asbestos.	1	2	3	4	5
List sources of information on asbestos.	1	2	3	4	5
2. The monograph addressed the objectives listed under "How to use this issue . . ."	1	2	3	4	5
3. I am more likely to ask patients questions regarding possible environmental exposure as a result of reading this issue.	1	2	3	4	5
4. Independent study was an effective teaching method for the content.	1	2	3	4	5
5. How much time (in minutes) was required to read this monograph and complete the posttest?	40	60	80	100	120

Comments: _____

POSTTEST: ASBESTOS

Circle all correct answers and transfer your answers to the answer sheet immediately following.

1. Which of the following people might encounter significant exposure to asbestos?
   a. workers in the construction trades
   b. persons eating vegetables grown on asbestos-contaminated soil
   c. boiler workers
   d. families of construction workers
   e. auto mechanics
2. Which of the following statements about asbestos are true?
   a. drinking asbestos-contaminated water results in pancreatic cancer
   b. fibers in the 5- to 10-micron range can be deposited in the lower regions of the lung
   c. chrysotile is the most commonly encountered type of asbestos
   d. fibers larger than 25 microns can reach the terminal bronchioles
   e. fragmentation of asbestos fibers into uncoated fibrils aids in excreting asbestos
3. The earliest signs of asbestosis may include
   a. radiologic findings of reticular fibrosis
   b. dyspnea on exertion
   c. cough
   d. pleural plaques
   e. hemosiderosis
4. Education, one of the foremost means of managing asbestos-related disease, should include warning patients to avoid
   a. smoking
   b. pulmonary infections
   c. traveling
   d. use of aspirin
   e. extreme temperature changes
5. Which of the following statements regarding asbestos exposure are true?
   a. asbestosis only occurs in patients with a history of prolonged exposure
   b. a chest X ray is the most sensitive indicator of asbestos-induced fibrosis
   c. the most common radiologic finding in exposed persons is pleural plaques
   d. the definitive test for diagnosing asbestosis is lung biopsy
   e. CT scanning should be used to screen all asbestos victims
6. Which of the following statements regarding asbestos are true?
   a. asbestos products remain in many older homes
   b. asbestos is no longer being used in the United States
   c. asbestos use was widespread primarily because of its low cost
   d. asbestos exposure does not occur today
   e. cigarette smoking does not affect asbestos-related diseases
7. The organs or systems that may be directly or indirectly affected by asbestos are
   a. lungs
   b. CNS
   c. liver
   d. cardiovascular
   e. immune
8. The differential diagnosis of fibrotic lung disease should include
   a. radiation
   b. pulmonary infections
   c. coal or silica dust
   d. sarcoidosis
   e. hemosiderosis

The **American Association of Occupational Health Nurses (AAOHN)**. AAOHN has approved this program for 1.2 contact hours. Applicant will receive the assigned code number in the award letter.

The **American Board of Industrial Hygiene (ABIH)**. ABIH has approved this program for 0.5 certification maintenance (CM) point per 3 Case Studies. The CM approval number is 2817.

To receive continuing education credit (CME or CEU), complete the following Posttest in the manner shown in the sample question below. **Circle all correct answers.**

Which of the following is known to precipitate migraine headaches?

(a.) fatigue
(b.) alcohol
c. grapefruit
(d.) sunlight
e. sleep

NOTE FROM NLM: TO RECEIVE CONTINUING EDUCATION CREDITS, YOU MUST RETURN A HARD COPY OF THE COMPLETED ANSWER SHEET AND EVALUATION TO THE CONTINUING EDUCATION COORDINATOR AT ATSDR. BE SURE TO INCLUDE YOUR NAME, ADDRESS, AND OTHER INFORMATION REQUESTED.

After you have finished the Posttest, please transfer your answers to the answer sheet and complete the evaluation on the lower half of that page. Mail the completed pages to:

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Your confidential test score will be returned with an indication of where the correct answers can be found in the text. Validation of earned CME credit and CEU will also be forwarded to participants, and their names, if requested, will be placed on the mailing list to receive other issues in the *Case Studies in Environmental Medicine* series.

A thorough medical and occupational history; a physical examination, including auscultation of the heart and lungs; chest X ray; and spirometry to assess possible restrictive and/or obstructive pulmonary disease may be indicated. Stool hemoccult testing is also advised.

(7) It would be prudent to have periodic evaluations including chest X ray and pulmonary function testing, and screening for colorectal cancer on a yearly basis.

(8) Parents often feel resentful that they have not been informed earlier of an asbestos hazard. A respected physician in the community is often able to put the risk of disease due to asbestos into perspective for such an audience. Before making public statements, however, it would be advisable to consult with state and local public health officials on the potential for asbestos exposure in local schools.

Sources of Information

More information on the adverse effects of asbestos and the treatment and management of asbestos-exposed persons can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: Asbestos Toxicity* is one of a series. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

Posttest and Credits

Continuing education credit is available to health professionals who use this monograph and complete the posttest. The criterion for awarding continuing medical education (CME) credits and continuing education units (CEU) is a posttest score of 70% or better.

The Centers for Disease Control (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians, and by the International Association for Continuing Education and Training (IACET) to sponsor continuing education units for other health professionals.

The Agency for Toxic Substances and Disease Registry, in joint sponsorship with CDC, is offering 1 hour of CME credit in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 hour of CEU for other health professionals upon completion of this monograph.

In addition, the series *Case Studies in Environmental Medicine* has been reviewed and is acceptable for credit by the following organizations:

The **American Academy of Family Physicians (AAFP)**. This program has been reviewed and is acceptable for 1 prescribed hour by the American Academy of Family Physicians. (Term of Approval: beginning January 1992.) For specific information, please consult the AAFP Office of Continuing Medical Education.

The **American College of Emergency Physicians (ACEP)**. Approved by the American College of Emergency Physicians for one hour per issue of ACEP Category I credit.

The **American Osteopathic Association (AOA)**. AOA has approved this issue for 1 credit hour of Category 2-B credit.

Answers to Pretest and Challenge Questions

Pretest

(a) The patient's symptoms are unlikely to be related to asbestos exposure. The patient's after-school activity for only 3 years is much less than the typical latency period for asbestos-associated diseases in workers. Asbestos levels measured in the general indoor air in schools also tend to be well below the OSHA permissible workplace level. A more likely cause of the boy's symptoms would be onset of bronchial asthma.

(b) The patient's potential exposure could place him at risk for future asbestos-related complications. Even low-level environmental asbestos exposures can eventually result in disease.

(c) The cousin's mesothelioma is unlikely to be related to his 3-year history of school custodial work. A number of cases of mesothelioma in long-term school custodians have been documented, however. In several recent studies, school custodians were also found to have asbestotic chest X rays. Exposure to airborne asbestos while working as a longshoreman is the more likely cause of the cousin's disease.

Challenge Answers

(1) If the pipe coverings are visibly in good condition and air sampling indicates no release of fibers, it is probably safer to leave them intact. Application of a substance to encapsulate the intact asbestos may be considered. If the pipe coverings are deteriorating, however, the family should seek professional advice from a qualified and licensed contractor specializing in asbestos abatement.

(2) The patient may be exposed to low levels of asbestos at home, school, and play. Asbestos materials adequately contained and not airborne are not likely to be a significant hazard, but asbestos does tend to be liberated from aging materials such as wall and ceiling insulation, or pipe and duct coverings. Asbestos-containing materials aggressively abraded may also release fibers. Power-buffing of asbestos-containing floor tiles is an example. The father's occupation suggests the patient could be receiving secondary asbestos exposure from dust brought home on his father's work clothes and person.

(3) Yes, workers exposed to asbestos can bring fibers home on their clothes, skin, and hair, inadvertently exposing others in the household.

(4) See (c) in the Pretest answers above.

(5) For the child described in the case study, the physician should clearly state that the child's symptoms are not likely to be attributable to asbestos, without unduly minimizing the possible long-term risks of asbestosis or cancer. The synergistic effects of smoking and exposure to other carcinogens should be discussed. If either or both parents smoke cigarettes, the child may be more likely to become a smoker himself, and thereby increase his risk of asbestos-related lung cancer. Also, parental smoking could expose the child to "second-hand" smoke.

(6) Yes, the father may be at increased risk for asbestos-related disease. Homes built before 1975 were typically constructed with asbestos-containing products. Removing or repairing these materials could liberate asbestos fibers that might be inhaled if appropriate respiratory protection is not worn.

Diagnosis

Craighead JE, Abraham JL, Churg A, et al. The pathology of asbestos-related diseases of the lungs and pleural cavities: diagnosis criteria and proposed grading schema. Report of the Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health. Arch Pathol Lab Med 1982;106:544-96.

Asbestos in Schools

American Academy of Pediatrics Committee on Environmental Hazards. Asbestos exposure in schools. Pediatrics 1987;79:301-5.

Mossman BT, Bignon J, Corn M, Seaton A, Gee JB. Asbestos: scientific developments and implications for public policy. Science 1990;247:294-301.

Carcinogenicity

Archer VE, Rom WN. Trends in mortality of diffuse malignant mesothelioma of pleura [Letter]. Lancet 1983;July 9:112-3.

Asbestos in water [Editorial]. Lancet 1981;2:132.

Chahinian AP, Pajak TF, Holland JF, et al. Diffuse malignant mesothelioma_prospective evaluation of 69 patients. Ann Intern Med 1982;96:746-55.

Craighead JE, Mossman BT. The pathogenesis of asbestos-associated diseases. N Engl J Med 1982;306:1446-55.

Davies D. Asbestos related diseases without asbestosis [Editorial]. Br Med J 1983;287:164-5.

Davies D. Are all mesotheliomas due to asbestos? [Editorial]. Br Med J 1984;289:1164-5.

Garrahan K. Mesothelioma: Has patient had contact with even small amounts of asbestos? [News]. JAMA 1987;257:1569-70.

Lemen R. Report on cancer risks associated with the ingestion of asbestos. Environ Health Perspect 1987;72:253-65.

Levine DS. Does asbestos exposure cause gastrointestinal cancer? Dig Dis Sci 1985;30(12):1189-98.

Owen WG. Mesotheliomal tumors and exposure to asbestos dust. Ann NY Acad Sci 1985;132:674-9.

Ross R, Dworsky R, Nichols P, et al. Asbestos exposure and lymphomas of the gastrointestinal tract and oral cavity. Lancet 1982;2:1118-20.

Walker AM. Declining relative risks for lung cancer after cessation of asbestos exposure. J Occup Med 1984;26:422-6.

Government Publications

Agency for Toxic Substances and Disease Registry. Toxicological profile for asbestos. Atlanta: US Department of Health and Human Services, Public Health Service, 1990.

Standards and Regulations

Workplace

The current OSHA standard for asbestos in the workplace is 0.2 fibers/cc of air as an 8-hour TWA.

In the 1930s, widespread evidence of asbestos-associated disease in workers was found. A standard for exposure was not established in this country until 1960 in selected industries; in 1971, it was extended industrywide. A 1968 British study judged that exposure to 2 fibers per cubic centimeter of air (fibers/cc) for the duration of a person's worklife would result in approximately a 1% risk of developing asbestosis, which was an underestimation. This estimate, nonetheless, led to the 1976 U.S. standard of 2 fibers/cc as a time-weighted average (TWA). Further study of carcinogenicity resulted in the U.S. standard of 0.2 fibers/cc (8-hour TWA), effective in 1986. The current level at which employers must take action to reduce employee exposure (action level) is 0.1 fibers/cc (8-hour TWA).

Environment

EPA's proposed MCL for asbestos in drinking water is 7 million fibers per liter of water.

The difficulties of controlling asbestos exposure in the workplace are paralleled in the general environment. EPA recommends "no visible emissions." In 1973, EPA banned spraying of asbestos in building interiors. Currently there is no regulation for asbestos in potable water. EPA's proposed maximum contaminant level (MCL) for asbestos in drinking water is 7 million fibers (larger than 10 microns in length) per liter of water.

The Asbestos in Schools Identification and Notification Act of 1982 requires that local education agencies 1) inspect for friable material, 2) analyze these materials for asbestos content, 3) post results and notify parents and employees if asbestos is found, and 4) maintain appropriate records. A recent study indicating that power-buffing and power-stripping of asbestos-tile floors in schools produces significant airborne-asbestos levels has prompted an EPA warning to school communities. Floor maintenance will henceforth be performed by hand to prevent the release of fibers.

Suggested Reading List

General

Council on Scientific Affairs. A physician's guide to asbestos-related disease. JAMA 1984;252:2593-97.

McCunney RJ. The patient with asbestos exposure. J Fam Pract 1986;22(1):73-8.

Nicholson WJ, Perkel G, Selikoff IJ. Occupational exposure to asbestos: population at risk and projected mortality--1980-2030. Am J Ind Med 1982;3:259-311.

Pendergrass HP, Snell JD, Carroll FE. Diseases related to asbestos exposure: historical perspective. South Med J 1987;80:1546-52.

Rubin AH. Common problems in asbestos-related pulmonary diseases. Am J Ind Med 1986;10:555-62.

Asbestosis is an irreversible pulmonary condition. Respiratory infections should be treated aggressively since they often prove fatal in patients with advanced fibrotic lung disease. Patients should be strongly advised to avoid all pulmonary irritants including cigarette smoke. Influenza and pneumococcal vaccines are warranted. In the later stages, pulmonary rehabilitation may be helpful. The patient should be advised to consult a physician when the first signs and symptoms of respiratory infection occur so that early treatment can be instituted.

Although most investigators consider the pleural plaques associated with asbestosis to be benign, they can result in pulmonary impairment. Patients with pleural asbestosis are also more likely to have or develop parenchymal asbestosis and should be followed appropriately. Patients should be informed that pleural plaques represent evidence of significant asbestos exposure.

Cancer

Mesothelioma

Patients with mesothelioma have a 1-year survival rate of less than 30%.

The prognosis for patients with mesothelioma is poor; they seldom live longer than 12 to 18 months after diagnosis. The 1-year survival rate of mesothelioma patients is less than 30%, and no efficacious treatment has been identified. Palliation and support are recommended.

Lung Cancer

Treatment of asbestos-associated cancer does not differ from treatment for cancers due to other causes.

Treatment of asbestos-associated cancer should include appropriate combinations of surgery, chemotherapy, and radiation, according to accepted surgical and oncological standards.

Challenge

(7) *If examination of the father of the child described in the case study is entirely normal except for bilateral pleural plaques, what follow-up will you recommend?*

_____

_____

(8) *As a concerned family physician, you become identified as a community resource on asbestos exposure and accept an invitation to speak at a Parent-Teacher Association meeting. What will you tell your audience?*

_____

_____

_____

Sputum Studies

Sputum studies are not useful for screening patients exposed to - asbestos.

Sputum inspection for asbestos fibers or ferruginous bodies has been advised, but most investigators now agree that the lack of sensitivity and specificity precludes their use for screening purposes. Sputum cytology remains useful as a diagnostic test for neoplasia and lung cancer, however.

Other Tests

Recent studies suggest that lymphocyte abnormalities (particularly T-cell) correlate with both asbestos-related malignancies and asbestosis. However, because these findings are in the early investigative stage, they are not clinically useful. There is no blood test that is useful for diagnosing asbestos-associated diseases.

Challenge

(6) Is the father (aged 50) of the patient described in the case study at risk of asbestos-associated disease; if so, what medical evaluation should be undertaken?

_____

_____

Treatment and Management

Patient education is an important factor in managing asbestos-associated disease.

Management of asbestos-associated diseases begins with patient education regarding smoking cessation and avoidance of pulmonary infections. Awareness of early symptoms of other neoplasms is important, including hoarseness, sores in the mouth, blood in the urine, blood in the stool, and gastrointestinal symptoms. Persons exposed to asbestos should be advised of the increased risk of lung cancer and the synergistic effects of cigarette smoking, although smoking does not affect the development of mesothelioma. Explaining environmentally related cancer risk is difficult because extrapolation of risk from workplace data is impossible in many cases. Maintaining a balance between appropriate concern and avoidance of undue alarm is the goal.

Follow-up of asymptomatic patients exposed to asbestos is recommended to facilitate early diagnosis and intervention. Periodic pulmonary function studies can be helpful in diagnosing early signs of asbestosis.

Asbestosis

Asbestosis patients should avoid pulmonary irritants and guard against lung infections.

Most pleural plaques are benign and require no specific treatment.

Radiographic Techniques

Radiographic results should not be used preferentially in diagnosing asbestosis.

The chest X ray is the basic tool for assessing asbestos-associated parenchymal and pleural disease. Radiographic findings may include interstitial fibrosis in the lower lung fields and thickening of both the parietal and visceral lung pleura. Parietal pleural thickening generally appears as a lobulated prominence of the pleura adjacent to the thoracic margin. Visceral pleural thickening is generally more diffuse and appears as interlobar fissure thickening on lateral films. A system has been proposed by the International Labor Organization for radiographic rating of the changes in pneumoconioses. The diagnosis of asbestosis should be made in the context of the overall clinical presentation and should include, but not emphasize, X ray findings. The association of pleural thickening and calcification enhances diagnostic accuracy; however, open lung biopsy is the only definitive diagnostic test for asbestosis.

The radiologic appearance of asbestos-induced lung cancer does not differ from that of other cancers. Asbestos-related malignancies predominantly involve the lower portion of the lungs, but they are not restricted to this location.

Computerized Tomography

CT scanning is too expensive for use as a screening tool, but may be helpful in certain cases.

Computerized tomography (CT) scanning is a particularly sensitive means of differentiating asbestos-related pleural plaques from soft-tissue densities. The technique is being used to diagnose other asbestos-associated abnormalities as well. Because it is considerably more expensive than standard X rays, CT scanning should not be considered a screening tool.

Pulmonary Function Testing

Small airway disease and restrictive defects are typical in nonsmoking patients with asbestosis; combined obstructive/restrictive pattern is more typical in smokers.

Nonsmoking patients with asbestosis typically have spirometric changes indicative of small airway disease and restrictive defects; smokers with asbestosis may have a combined obstructive/restrictive pattern. Small airway disease is a common early finding and is reflected in a 25% to 74% reduction of forced expiratory flow rates. This change may reflect early fibrosis in the peribronchiolar areas or inflammatory changes. Restrictive defects are observed as a reduction in forced vital capacity. Because such reduction may also occur in obstructive airway disease, an apparent combined pattern of restrictive and obstructive disease should be followed up with further pulmonary studies including carbon monoxide diffusion capacity and static lung volumes. True restrictive disease generally manifests as a decrease in total lung capacity with normal or residual volume, which can be determined using both the plethysmographic and helium dilution methods.

costophrenic angles and extends diffusely up the chest walls. If advanced, visceral pleural thickening can be associated with dyspnea and restrictive changes on pulmonary function tests.

A patient with asbestosis commonly develops fatigue, weight loss, and insidious onset of dyspnea on exertion. As the disease progresses, the dyspnea worsens. A dry cough typically occurs, but a productive cough, even in a nonsmoker, is not uncommon. Patients often describe a "tight" feeling in the chest. The interstitial disease is radiographically demonstrated as a reticular fibrosis located predominantly in the lower lung fields. Radiologic evidence is often not present until at least 5 years after exposure.

Fibrosis found symmetrically in the lower aspects of both lungs is typically caused by asbestos. Fibrotic lung disease due to asbestos inhalation is often associated with pleural plaque formation, which eliminates other etiologic possibilities such as drugs, radiation, sarcoidosis, collagen vascular disorders, Goodpasture's syndrome, hemosiderosis, idiopathic pulmonary fibrosis secondary to lung infections, and inhaled silica, coal dust, or organic dusts.

Lung Cancer

Asbestos-associated lung cancers produce the same symptoms as cancers due to other etiologies.

Lung cancer caused by asbestos exposure cannot be differentiated from cancer caused by other environmental factors. The differential diagnosis of lung cancer in an asbestos-exposed patient should include other possible etiologies such as exposure to cigarette smoke, arsenic, chloromethyl ethers, chromium, nickel, and ionizing radiation. Clubbing of the distal phalanges or cyanosis of the nail beds may be present.

Mesothelioma

The latency period for mesothelioma is 20 years or more, but the onset of symptoms is sudden.

Both pleural and peritoneal mesotheliomas may be seen in asbestos-exposed patients. These tumors are rapidly invasive. Although onset of mesothelioma is not sudden, symptoms of the disease may be. Peritoneal mesotheliomas are more difficult to diagnose by noninvasive means than pleural occurrences. They are frequently detectable as an expanding "doughy" feeling on abdominal palpation. Mesothelioma is seldom associated with etiologies other than asbestos exposure.

Laboratory Tests and Special Procedures

Chest X ray and pulmonary function tests are important procedures in diagnosing asbestos-associated disease.

Established tests and procedures helpful in diagnosing asbestos-associated disease include radiographic techniques, pulmonary function tests, and possibly computerized tomography scanning. Neither sputum studies nor blood chemistry studies are useful in diagnosing asbestos-associated disease in the clinical setting.

Challenge

(4) *Is the mesothelioma of the patient's cousin likely to be related to his school custodial work? Explain.*

_____

(5) *How will you explain the patient's potential health risks to his mother?*

_____

_____

Clinical Evaluation

History and Physical Examination

Dry rales, auscultated in the midaxillary line, are the most common lung findings associated with asbestosis.

The medical evaluation of workers exposed to asbestos includes a thorough medical and occupational history, physical examination, chest X ray, and pulmonary function tests. The same protocol has been recommended for evaluating an asymptomatic patient with a history of asbestos exposure. Pertinent historical information includes the source, intensity and duration of exposure, time elapsed since first exposure, and work history of household members. Asbestos accumulates in the body, and even relatively minor exposures may be important.

The physical examination should focus primarily on the patient's lungs, and particular attention should be paid to pulmonary auscultation. Fine inspiratory rales in the posterior and posterolateral lung bases, audible on deep inspiration, may be the earliest sign of interstitial fibrosis. Generally, however, a chest X ray is more sensitive.

Examination should also assess stigmata of other diseases that may confound the diagnosis of asbestosis. For instance, rheumatoid arthritis is sometimes associated with interstitial fibrosis. Chest-wall configuration, evidence of thoracic surgery, and cardiac status may also affect the diagnosis.

Signs and Symptoms

Significant clinical syndromes include asbestosis, lung cancer, and mesothelioma.

Asbestosis

Progressive dyspnea on exertion is a common symptom of asbestosis.

The most common finding in asbestos-induced pulmonary disease is pleural thickening, often manifested as discrete pleural plaques. Pleural plaques can be seen as radiologic bilateral images of hyalin scar formation on either the visceral or parietal pleural surfaces. Involvement of the parietal pleura rarely is associated with symptoms. Visceral pleural thickening often involves blunting of the

Lung cancers of most cell types (except alveolar cell) have been associated with asbestos exposure. Adenocarcinoma of the lung occurs more often in asbestos-exposed persons than in nonexposed persons. Asbestos-associated lung cancer tends to occur in the lower lung fields, although not exclusively.

Other Carcinogenic Effects

Increased incidence of gastrointestinal cancers has been reported among asbestos workers.

The consequences of ingesting asbestos fibers are controversial.

Some mortality studies of asbestos workers have revealed small increases in the incidence of death from cancer at one or more extrathoracic sites, including the kidneys and gastrointestinal system - notably the esophagus, stomach, colon, and rectum. Presumably these cancers are due to swallowing asbestos fibers. In contrast, other epidemiologic studies have not detected statistically significant associations between asbestos ingestion and extrathoracic cancers. Various researchers and regulatory groups have reviewed the weight of evidence and have not been able to reach a consensus on the effects of ingested asbestos fibers. Whether gastrointestinal neoplasms may be induced by ingesting asbestos-contaminated drinking water remains unproven. In humans, asbestos bodies have been identified in extrapulmonary tissues including tonsils, thoracic and abdominal lymph nodes, pleura, peritoneum, liver, spleen, kidneys, adrenals, small intestine, pancreas, and bone marrow, as well as the lungs.

Cardiovascular Effects

Cardiovascular effects are secondary to pulmonary fibrosis.

Fibrosis of the lung can lead to increased resistance to blood flow through the capillary bed, resulting in cor pulmonale. This condition may also occur with less severe fibrotic disease, especially if chronic obstructive lung disease is simultaneously present, as commonly seen in cigarette-smoking asbestos workers. Pulmonary hypertension may occur before decreased respiratory function is clinically detectable.

Immunologic Effects

Immunologic abnormalities have been noted in persons with asbestosis.

Immunologic abnormalities have been observed in asbestos workers with clinical signs of asbestosis and have also been reported in persons environmentally exposed. Despite some variability, most studies indicate that cell-mediated immunity can be depressed in workers who have radiologic evidence of asbestosis. Autoantibodies (rheumatoid factor, antinuclear antibodies) are typically present in these workers. Caplan's syndrome (the coexistence of pneumoconioses with rheumatoid changes) also has been noted in asbestos workers, although it is more common in coal miners and workers with other pneumoconioses. The implications of these immunologic changes are difficult to assess, but they are of special concern because depressed immune function might be a factor in the etiology of asbestos-induced cancer.

Pleural effects can occur even in the absence of parenchymal asbestosis. The incidence of pleural abnormalities in persons employed in asbestos-related occupations can be high (20% to 60%). Asbestos effects on the pleura include plaques (with and without calcification), diffuse pleural thickening, and effusions. Pleural plaques are oval areas of acellular collagen deposits, usually located bilaterally on the inferior and posterior surfaces of the pleura. People in contact with work clothes of asbestos workers or with asbestos-containing household products have developed pleural abnormalities. An asbestosis prevalence of 11% in wives, 8% in sons, and 2% in daughters was reported in families of asbestos-exposed shipyard workers.

Pleural plaques are not lung cancer precursors, although persons with pleural plaques have an increased incidence of lung cancer. Migration of inhaled asbestos to the pleura is the most likely cause of plaques. Pleural thickening can lead to decreased ventilatory capacity, probably because of restrictive effects. These effects are most commonly seen with extensive involvement of the visceral pleura, which is observed radiologically as diffuse pleural thickening.

Mesothelioma

Mesothelioma is a signal tumor for asbestos exposure and can appear after relatively low-level exposures.

Unlike bronchogenic cancer, mesothelioma risk is not affected by cigarette smoking.

Mesotheliomas are tumors arising from the thin membranes that surround internal organs. Pleural and peritoneal mesotheliomas are rare in the general, unexposed population and are indicators of asbestos exposure. Although all asbestos types can cause mesothelioma, several studies have suggested that, in humans, the amphibole mineral form may be more likely to induce mesothelioma than the serpentine form.

The dose appears to be lower for asbestos-induced mesothelioma than for pulmonary asbestosis or lung cancer. An extremely short exposure period may be sufficient to cause this rare tumor. However, there is typically a long latency period. Latency periods have been up to 57 years, although more intense exposures can result in latencies as short as 20 to 30 years. Some studies have indicated that risk of mesothelioma from a given level of asbestos exposure depends primarily on the elapsed time since exposure, with risk increasing dramatically after a lag period of about 10 years.

An estimated 1500 cases of mesothelioma per year occur in the United States (compared to an average of 130,000 cases of lung cancer per year, mostly due to smoking). Data on death rates from pleural or peritoneal mesotheliomas over the past 10 to 20 years indicate that mesotheliomas are increasing in males over 65 years of age who have an occupational history of asbestos exposure. Unlike asbestos-related bronchogenic cancer, mesothelioma risk does not appear to be influenced by smoking.

Lung Cancer

Latency for lung cancer is 10 to 30 years or more.

It is unclear whether a threshold asbestos dose exists for lung cancer.

There is little doubt that all types of asbestos can cause lung cancer. A latency period of 10 to 30 years or more exists between the onset of asbestos exposure and occurrence of the tumor. Whether asbestos exposure will lead to lung cancer depends not only on cumulative exposure, but also on other underlying lung cancer risks.

Physiologic Effects

Asbestos primarily affects the respiratory system. The immune and cardiovascular systems, and possibly the GI system, are also affected by asbestos exposure.

The respiratory, immunologic, cardiovascular, and gastrointestinal systems may be adversely affected by asbestos inhalation and by ingestion subsequent to mucociliary removal from the respiratory tract. Skin nodules from handling asbestos-containing materials may also occur.

Immunologic abnormalities such as increased concentrations of auto-antibodies and depressed lymphocyte responsiveness (discussed later in this document) are usually mild or absent in persons who have not developed clinical signs of asbestosis. Cardiovascular effects are secondary to pulmonary changes. Fibrosis in the lung can lead to increased resistance to blood flow through the pulmonary capillary bed, resulting in pulmonary hypertension and compensatory hypertrophy of the right heart.

No deaths due to acute exposure to asbestos have been reported. However, delayed death due to asbestosis and cancer from chronic inhalation exposure has occurred. The risk of developing asbestos-associated disease continues even after exposure has ceased.

Respiratory Effects

Asbestos exposure may result in asbestosis, mesothelioma, or carcinoma.

Inhalation of asbestos fibers may cause parenchymal and pleural asbestosis, mesothelioma, and carcinoma. All four syndromes can be present in a patient. Exposure to other carcinogens, dose and duration of exposure, individual susceptibility, and elapsed time since initial exposure all may play a role in disease development. Chronic low-level asbestos exposure has been associated with lung cancer, mesothelioma, and pleural diseases, including pleural asbestosis; higher doses are more likely to produce parenchymal asbestosis. Smoking and exposure to other toxins increase the risk of asbestos-associated lung cancer.

Asbestosis

Asbestosis is pulmonary interstitial fibrosis of the pleural or parenchymal tissue.

Pleural plaques have not been shown to be premalignant.

Inhalation of asbestos fibers can lead to a characteristic pneumoconiosis, or diffuse interstitial fibrosis, termed asbestosis. Either heavy exposure for a short time or lower-level exposure over a longer period may result in asbestosis; cases have resulted from intense exposure of 1 day's duration. The disease can affect the lung parenchyma or pleural tissue. Clinical manifestations typically appear 20 to 40 years after onset of exposure. Radiologic changes can occur before 20 years, however.

Asbestosis patients typically have elevated levels of antinuclear antibody and rheumatoid factors and a progressive decrease in total lymphocyte count with advancing fibrosis. Self-perpetuating host responses may be affecting the progression of fibrosis even after exposure ceases.

Biologic Fate

A significant proportion of inhaled asbestos fibers may be retained in the lungs.

The size and shape of asbestos fibers affect the lungs' ability to effectively remove them.

The primary route of asbestos entry into the body is through inhalation. Ingestion of asbestos fibers can occur from drinking contaminated water or after mucociliary clearance from the lungs. The fate of ingested asbestos is still being debated. Asbestos fibers may also lodge in the skin.

Generally, only particles between 0.5 and 5 microns in diameter with a length-to-width ratio of 3:1 will be deposited in the respiratory regions of the lung (alveoli and terminal bronchioles). Larger particles tend to be filtered out in the upper airway and nasopharynx. Smaller fibers tend to remain suspended in the inspired air, and the majority are exhaled. However, asbestos is an exceptional substance: fibers ranging from 5 to 10 microns in diameter can also penetrate to the lower respiratory regions of the lung, where they may have destructive effects.

The fibrous nature of asbestos renders the lungs' defense mechanisms ineffective. Smaller, nonfibrous particles are normally engulfed by macrophages and removed by lymphatic or mucociliary mechanisms. Attempts by the macrophages to engulf fibers can lead to eventual deposition in various tissues of ferrous material in a drumstick configuration called a ferruginous body (asbestos body).

Asbestos fibers can also penetrate to the terminal bronchiolar level and enter the peribronchiolar space, resulting in a fibrogenic response. Because the fibers concentrate in the lower lung fields, there is a tendency for fibrosis to occur first in the lungs' bases, and for pleural effects to be confined to the lower two-thirds of the thorax. Fibrosis results from persistent release of inflammatory mediators such as lysozymes, interleukins, and fibroblast growth factors at the site of asbestos fiber penetration and deposition.

Data do not clearly relate GI tumors or peritoneal mesotheliomas to direct ingestion of asbestos fibers, although persons exposed to asbestos by inhalation have been reported to have a twofold greater risk of colorectal cancer than unexposed persons. Some investigators believe this malignancy is caused by fibers removed from the lungs' upper respiratory regions by ciliary mechanisms and then swallowed. Most reports suggest that ingested asbestos is excreted with the feces. Asbestos bodies have been identified within some human specimens of colorectal adenocarcinomas. Several animal studies have revealed that asbestos fibers are capable of penetrating the GI tract.

Electron microscopy reveals that fibrils result from longitudinal and cross-sectional fragmentation of asbestos fibers. A single asbestos fiber can fracture into hundreds of sub-microscopic fibrils. Research indicates that these uncoated fibrils may be the form that migrates into the peritoneal and pleural spaces.

considerable. Carpenters, utility workers, electricians, pipefitters, steel mill workers, sheet metal workers, boilermakers, and laborers are at risk of exposure to asbestos through construction materials, insulation coverings of pipes, boilers, industrial furnaces, and other sources. Mechanics working with brake and transmission products also may be exposed to asbestos.

Secondary exposure occurs when fibers released to the air are inhaled by persons not directly handling asbestos. For example, 4 to 5 million shipyard workers were exposed when a relatively small number of insulation workers applied asbestos to ships' pipes and hulls. Domestic and environmental asbestos exposures may also occur indirectly. Asbestos-related diseases have occurred in family members whose only contact was dust from an exposed worker's clothes. Similar diseases were also found in persons who grew up within one-half mile of an asbestos factory.

Cigarette smoking and exposure to other carcinogens greatly increase the risk of asbestos-associated lung cancer. A comparison of the experiences of 17,800 asbestos insulation workers with matched controls showed that asbestos workers who did not smoke suffered 5 times the number of lung cancer deaths than controls who neither smoked nor worked with asbestos (55 deaths per 100,000 man-years for asbestos workers who did not smoke compared with 11 deaths per 100,000 man-years for controls who were neither asbestos workers nor smokers). Persons who smoked but did not work with asbestos had a death rate of 122 per 100,000 man-years; and among persons with both exposures (asbestos and cigarette smoking), 601 deaths occurred per 100,000 man-years. There is evidence that cigarette smoking in asbestos workers is also associated with increased risk of cancer of the esophagus, oropharynx and buccal cavity, and larynx. Cancers of the stomach, colon-rectum, and kidney, however, do not appear to be synergistically affected by smoking and asbestos exposure since smoking and nonsmoking asbestos workers suffer equal incidences of these health effects. While cancer, when established, may be irreversible, cancer risk is reversible. Data indicate that risk diminishes when smoking ceases.

Challenge

(2) *On questioning the mother, you learn that the father of the boy described in the case study is a master carpenter who specializes in restoring Victorian-style homes. What are the potential sources of asbestos exposure for the child?*

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(3) *The spouse of the mother's cousin is reportedly in good health. Should she be screened for asbestos-related disease? Explain.*

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Indoor air may become contaminated with fibers released from building materials, especially if they are damaged or crumbling. Common sources in homes are sprayed asbestos ("cottage cheese") ceilings, pipe insulation, boiler coverings, wallboard, and floor and ceiling tiles. Homeowners should not undertake repair or removal of asbestos-containing materials without professional guidance or services.

Although measurable asbestos levels in schools are usually 1000 times below the permissible exposure limit (PEL) for work environments, public concern has led to widespread removal and abatement programs. However, some facilities have higher levels of airborne asbestos after removal than before, indicating that removing asbestos improperly can be more hazardous than leaving it in place.

Street dust may contain fibers from brake linings or crushed asbestos-containing rock used in road construction. Fibrous tremolite, the asbestos commonly found in talc, has also been found in play sand.

Drinking water supplies may become contaminated with asbestos from erosion of natural land sources, discarded mine and mill tailings, asbestos cement pipe, and from disintegration of other asbestos-containing materials transported via rain. Most water supply concentrations are less than 1 million fibers per liter but in some cases have exceeded 100 million fibers per liter. The U.S. Environmental Protection Agency's (EPA) proposed maximum contaminant level for asbestos in drinking water is 7 million fibers (larger than 10 microns in length) per liter.

Challenge

(1) *Additional information for the case study: The patient and his family live in a home built in 1955. Pipes in the basement are covered with asbestos insulation. Should the family consider the removal of all asbestos pipe coverings in their home? Explain.*

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Who's at Risk

Workers in the construction trades are most heavily exposed to asbestos.

Spouses and family members can be exposed through asbestos dust on worker's skin and work clothing.

Cigarette smoke increases the risk of asbestos-associated lung cancer.

In the past, asbestos exposure was associated mainly with mining and milling of the raw material and with workers engaged in product manufacture. Since industrial use has decreased over the last 40 years, these occupational exposures have declined. Today, most exposures occur during repair, renovation, removal, and maintenance of asbestos that was installed years ago. The number of new exposures to the general population from in-place asbestos, however, may be greater in number than the exposures to all earlier workers combined.

Exposure Pathways

Asbestos exposure occurs primarily through inhalation of fibrous dust.

Insulating materials produced prior to 1975 commonly contain asbestos.

Asbestos is a generic term for a group of six naturally occurring fibrous minerals. The basic unit of asbestos-class minerals is the silicate combined in varying proportions with magnesium, iron, calcium, aluminum, and sodium or trace elements.

There are two major classes of asbestos: serpentine, which contains a magnesium silicate called chrysotile, and amphiboles, which represent a small portion of the world's asbestos consumption and include crocidolite, amosite, anthophyllite, and tremolite. Chrysotile, the sole member of the serpentine group, accounts for 93% of the world's asbestos use.

Asbestos has been used in over 3000 products due to its high tensile strength, relative resistance to acid and temperature, and its varying textures and degrees of flexibility. It does not evaporate, dissolve, burn, or undergo significant reactions with other chemicals, which makes asbestos nonbiodegradable and environmentally cumulative.

Although many applications have been phased out of production, uses of asbestos have included the following:

Commercial

Boilers and heating vessels
Cement pipe
Clutch, brake, transmission components
Conduits for electrical wire
Corrosive chemical containers
Electric motor components
Heat protective pads
Laboratory furniture
Paper products
Pipe covering
Roofing products
Sealants and coatings
Textiles (including curtains)

Homes and Buildings

Duct insulation
Fire protection panels
Fireplace artificial logs or ashes
Furnace insulating pads
Fusebox liners
Heater register tape and insulation
Joint compounds
Patching plaster
Pipe or boiler insulation
Sheet vinyl or floor tiles
Shingles
Textured acoustical ceiling
Underlayment for sheet flooring

Asbestos fibers may result from mining, milling, and weathering of asbestos-bearing rock, and from the manufacture, wear, and disposal of asbestos-containing products. Because of the widespread use of asbestos, its fibers are ubiquitous in the environment.

Although bans and voluntary phaseouts have contributed to declining production of asbestos since the early 1970s, it is still used in construction materials, mostly asbestos cement products. Building insulation materials manufactured since 1975 may no longer contain asbestos; however, products made or stockpiled before the ban remain in many homes.

Case Study

A 10-year-old boy with shortness of breath and recent asbestos exposure

A 10-year-old boy is seen at your office with a chief complaint of shortness of breath. Exertional dyspnea has been present for the previous month and is associated with intermittent dry cough. The patient has no associated fever, chills, or chest pain. Chart review indicates no history of asthma or other pulmonary disease, although the patient has been seen several times for "hay fever."

The patient is accompanied by his mother, who appears quite anxious. The mother emotionally relates that her 65-year-old cousin has recently been diagnosed with mesothelioma and is dying. Furthermore, he had been a custodian at the patient's school for the previous 3 years, after retiring from his career as a longshoreman. His work at the school involved general cleanup and boiler room maintenance. The mother is afraid that her son's dyspnea and cough are related to asbestos exposure at the school and that he may be developing mesothelioma since he often helped his cousin after school. Recent asbestos removal in the school has increased the mother's concern.

On physical examination, the patient is in no acute distress. Respirations are unlabored. Lung auscultation reveals a diffuse, expiratory wheeze. Spirometry performed in the office shows an FVC of 95% of predicted value and an FEV_1 of 88% of predicted value with an FEV_1/FVC of 70%. The remainder of the examination is within normal limits. A chest X ray is normal.

$\mathcal{P}_{\text{retest}}$

(a) Discuss whether the patient's symptoms are related to asbestos exposure.

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(b) Is the patient at risk for future disease? Explain.

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(c) Can the cousin's mesothelioma be related to his work as a custodian in the school? Explain.

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Answers to the Pretest can be found later in this document.

How to use this issue. . .

This issue begins with a composite case study that describes a realistic encounter with a patient. This description is followed by a pretest. The case study is further developed through Challenge questions at the end of each section. To fully benefit from this monograph, readers are urged to answer each question when it is presented. (Answers to the Pretest and Challenge questions are found toward the end of this document.) The monograph ends with a posttest, which can be submitted to the Agency for Toxic Substances and Disease Registry (ATSDR) for continuing medical education (CME) credit or continuing education units (CEU). See the end of this document for further instructions on how to receive these credits.

The objectives of this monograph on asbestos are to help you:

- **Explain why asbestos may be an acute and chronic health hazard**
- **Describe the known factors contributing to asbestos toxicity**
- **Identify potential environmental or occupational sources of exposure to asbestos**
- **Identify evaluation and treatment protocols for persons exposed to asbestos**
- **List sources of information on asbestos**

Contents:

Case Study
Pretest
Exposure Pathways
Who's at Risk
Biologic Fate
Physiologic Effects
Clinical Evaluation
Treatment and Management
Standards and Regulations
Suggested Reading List
Answers to Questions
Sources of Information
Posttest and Credits

This issue is prepared with the assistance of those who share a common concern for physician education, public health, and the environment, including the following organizations: American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP), American College of Occupational and Environmental Medicine (ACOEM), American Medical Association (AMA), Association of State and Territorial Health Officials (ASTHO), and the Society of Teachers of Family Medicine (STFM). Final responsibility for the contents and views expressed in this monograph resides with ATSDR.

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Case Studies in Environmental Medicine

Asbestos Toxicity

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Environmental ALERT . . .

Although asbestos has been banned from use in many products, it will remain a public health concern well into the 21st century.

Intact asbestos sources in the home release few fibers and should be left undisturbed. Damaged or crumbling materials should be repaired or removed only after receiving professional advice.

Asbestos exposure is associated with asbestosis, mesothelioma, and lung cancer, and may cause cancer at extrathoracic sites.

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. See Posttest section of this document for more information about continuing medical education credits and continuing education units..

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Case Studies in Environmental Medicine

Asbestos Toxicity

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This case study and monograph have been edited for inclusion in the TEHIP Gopher. The pagination and layout of this document differ from that of the original case study as published by ATSDR because of data conversion. For example, **summary highlights**, appearing adjacent to relevant text in the ATSDR publication, are placed before the text to which they refer in the TEHIP gopher version. There may be other differences between this version and the official document.

The official ATSDR hard copy document is available from:

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Agency for Toxic Substances and Disease Registry
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